Narrow QRS tachycardia with atrial and ventricular cycle length wobbling – What is the mechanism?

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Case Presentation

A 41 years old lady presented to us with drug refractory narrow QRS tachycardia. Echocardiography revealed normal left ventricular systolic function. 12 lead electrocardiography (ECG) showed long RP (RP>PR) narrow QRS tachycardia. Radiofrequency catheter ablation was performed after obtaining informed consent. Intracardiac electrograms and 12 lead ECG were continuously monitored using Workmate Claris system (Abbott, Plymouth, MN). Upper limb Venous angiography showed dilated coronary sinus with left sided superior vena cava. Basal AH and HV intervals were 85ms and 44ms respectively. Patient had spontaneously inducible tachycardia (Fig 1B) which could be terminated by intravenous adenosine. ECG showed narrow QRS tachycardia with variation in both atrial and ventricular cycle length (Fig 1A). Intracardiac electrogram during tachycardia showed variation in H-H, A-A, V-V and HA intervals (Fig 2)

1. What is the mechanism of the tachycardia onset?
2. What is the substrate for re-entry?
3. What could be the reason for the variation in A-A, H-H, H-A and V-V intervals?

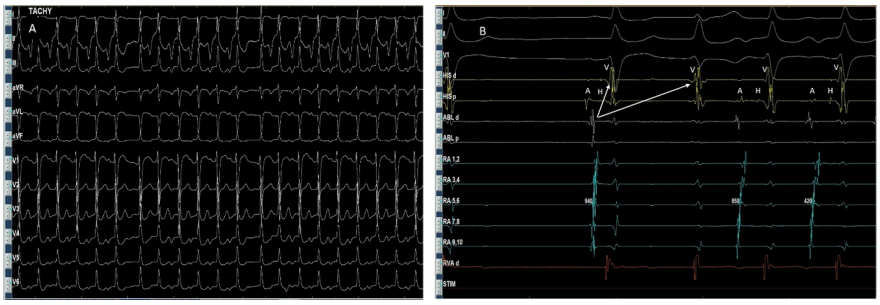

Fig 1: A -12 lead ECG showing narrow QRS tachycardia with varying RP interval. B – Electrogram showing the spontaneous initiation of tachycardia. Single atrial impulse has got conducted through both fast and slow pathways to produce 1:2 AV response followed by initiation of tachycardia. His d and His p – His electrograms – distal and proximal, ABL d and P – Ablation catheter distal and proximal, RA 1-10 – Coronary sinus electrograms, RVA d and p – RV electrogram distal and proximal

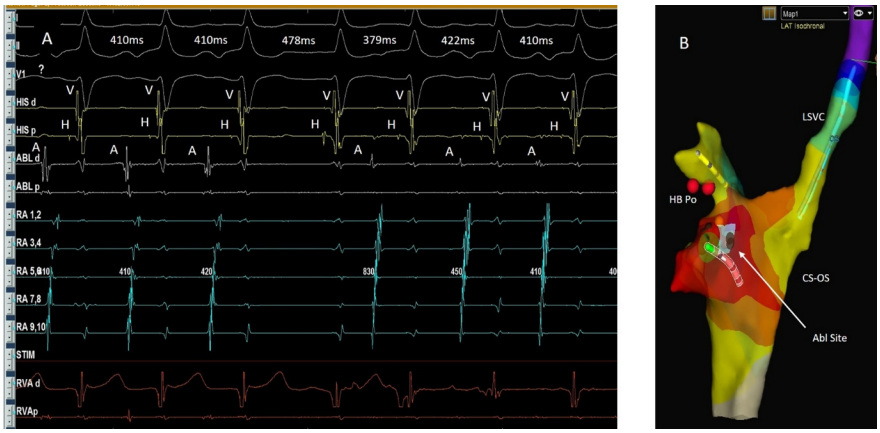

Fig 2: A – Intracardiac electrogram showing variation in A-A, H-H, H-A and V-V intervals due to decremental conduction in upper common pathway and antegrade slow pathway (S1). B- Electroanatomic map showing dilated coronary sinus ostium (CS-OS) with left SVC (LSVC) and the site of earliest atrial activation (white spot). His d and His p – His electrograms – distal and proximal, ABL d and P – Ablation catheter distal and

proximal, RA 1-10 – Coronary sinus electrograms, RVA d and p – RV electrogram distal and proxiamal, His po- His potential

Commentary:

12 lead ECG showed narrow QRS tachycardia with retrograde VA wenkebaching, hence Atrio-ventricular reciprocating tachycardia (AVRT) and atrial tachycardia (AT) could be excluded. Intracardiac electrogram during spontaneous induction showed classical 1:2 AV nodal conduction followed by initiation of tachycardia (Fig 1B). Several features favor AV nodal re-entrant tachycardia (AVNRT) in this case: VA dissociation, response to adenosine, 1:2 AV nodal conduction, concentric atrial activation pattern and fixed HV interval (44ms) during tachycardia. The possibility of automatic junctional tachycardia was excluded based on response to premature atrial extrastimuli and intravenous adenosine. Intracardiac electrogram showed variation in A-A, V-V, H-H and HA intervals. Atypical AVNRT of slow – slow type with decremental conduction of upper common pathway (UCP) could explain the variation in A-A and H-A intervals (Fig 3A, B and C). The H-H and V-V interval variations could be explained by presence of multiple slow pathways as below

1. Three slow pathways with different electrophysiological properties
2. Initial re-entry between slow pathway-1 (S1-antegrade) and slow pathway-2 (S2-retrograde) at a cycle length of 410ms with concealed conduction into slow pathway-3 (S3) and decremental conduction in upper common pathway (fig 3A)
3. Decremental conduction of S1 resulting in gradual prolongation of H-H interval followed by block which favored the transient re-entry between S3 (antegrade) and S2 (retrograde) (cycle length 379ms) and subsequent resumption of S1 conduction (fig 3B & C)

*Fig 3: A – Atypical slow- slow AVNRT with re-entry between S1 and S2 with concealed conduction into S3. Upper common pathway showed decremental conduction. B – Transient block in S1 due to decremental conduction favored re-entry between S3 and S2 with concealed conduction into S1. C – Ladder diagram showing the same phenomenon. The decremental conduction in S1 prolonged H-H/V-V interval from 410ms to 478ms. S3 could conduct in the next beat with H-H interval of 379ms as S1 was transiently blocked. In the subsequent beat S1 conduction resumed with minimal prolongation of H-H interval due to concealed conduction. Atrial conduction occurred decrementally through upper common pathway (UCP). S1,2,3 – slow pathways 1,2 and 3, F – fast pathway, LCP -Lower common pathway*The possibility of two antegrade slow pathway conduction has been described previously as a cause for cycle length alteration during AVNRT[1]. Multiple slow pathways are required for the initiation of re-entrant tachycardia after 1:2 AV response which otherwise would not have induced. In our patient the cycle length alteration was noted in both atrium (A-A) and ventricle (V-V). This could be explained by the rare combination of decremental conduction in both upper common pathway and antegradely conducting slow pathway (S1). The second pathway (S2) has different electrophysiological properties (conduction velocity and refractory period) as evidenced by the intracardiac electrogram and it is unlikely to be a fast pathway as the HA interval is long. The fourth beat in the figure 2C has slightly longer cycle length (422ms) due to concealed conduction of previous impulse into S1. These findings emphasize the fact that both atrium and the ventricle are not the part of circuit in AVNRT. Electroanatomic mapping (ENSITE Velocity, Abbott, Plymouth, MN) confirmed earliest atrial activation at coronary sinus ostium (fig 2B) Slow pathway was ablated (medium curve catheter; 60W, 600C) which rendered the tachycardia non-inducible. Since there were three slow pathways involved in this patient, this rare variety could be labelled as atypical slow-slow-slow AVNRT. The mechanism of re-entry in typical as well as atypical AVNRT remains elusive[2]. There has been electrophysiologic evidence of multiple superior atrial inputs to the AV node[3]that could explain multiple sites of early atrial activation during tachycardia. Cycle length alternans can occur during AVNRT due to either antegrade conduction via two slow pathways or junctional bigeminism[4]. The decremental conduction properties of antegrade slow pathway (S1) and upper common pathway were the reasons for variation in A-A, H-H, H-A and V-V intervals in our case of atypical slow-slow-slow AVNRT.**References**

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